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MEMO

TO: United States House of Representatives: Michigan
United States Senate: Michigan

FROM: Michelle Shafer RN, BA, CBIS, Medical Case Manager; Testimony

RE: Michigan House Bill No. 4936

DATE: October 13, 2011

In the proposed matter of ending Michigan No-Fault medical coverage for catastrophic injury: traumatic brain and spine injury, and transferring patient insurance coverage to State Medicaid. Medical documentation and support that this bill promotes a significant inability to provide reasonable and necessary medical care to a specialized patient population.

The catastrophic traumatic brain and spine injury patient population:

- Is not capable of 100% recovery; they are physically, mentally and emotionally altered for the duration of their lifetime and require lifetime medical care and treatment.

- Suffer white matter damage to pain pathways in the central nervous system causing atypical neurogenic pain (burning, stinging, stabbing, hot, cruel, gnawing in type and significant and disabling spasm) with higher pain severity ratios on the analog pain scale (0=no pain, 10=severe incapacitating pain). And, as such, do not respond to traditional pain treatment modalities; anti-inflammatory drugs and first line pain receptor medications and opiates and traditional physical therapy, and, in fact, symptoms can be exacerbated by traditional physical therapy techniques.

- Require unique pain treatment modalities:
    - Layered, time released opiates: oral Oxycontin, Fentanyl, Percosette
      Topical patch layering: Fentanyl
      Intrathecal baclofen, hydromorphone, fentanyl, clonidine

        - Implanted devices for pain control:
          Spinal Cord Stimulator with medical maintenance
          Intrathecal Pain Pump with monthly syringe medications

MICHIGAN MEDICAID DOES NOT COVER / PAY THE ABOVE TREATMENT MODALITIES

References attached:

Zasler, N. et al. Brain Injury and Pain Management, Journal of Head Trauma Rehabilitation, Vol 19, No. 1 (Jan 2004)

Oaklander AL, Rissmiller JG, et al. Evidence of focal small fiber axonal degeneration in complex regional pain syndrome-1. Pain, February 2006, vol. 120, pp 235-243.

D.R. Rutgersa, F. Toulgoata, J. et al. White matter abnormalities in Mild Traumatic Brain Injury: A diffuse tensor imaging study. American Journal of Neuroradiology 29:514-519, March 2008

Respectfully submitted,

Michelle L. Shafer RN, BA, CBIS, CM

Special Issue:
Brain Injury & Pain Management
Journal of Head Trauma Rehabilitation
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Preface

Traumatic brain injury (TBI) generates a broad range of disabling problems and impairment. Among the most challenging concomitants is that of pain. Because trauma to the brain is frequently accompanied by trauma to other bodily systems, it should not be surprising that pain is a clinical issue with various presentations at various stages posttrauma in a high number of survivors of TBI.

Given the complexity of both central and peripheral pain generators and perpetuators, as well as predisposing and reactive psychoemotional sequelae, management of TBI with comcomitant pain is often steeped in misconceptions, myths, misdiagnosis, and mistreatment.

Appropriate management of persons with TBI clearly requires an understanding of the causes of pain in this patient population in acute as well as postacute settings and a comprehensive knowledge base regarding treatment options for the variety of pain conditions that may present in these individuals. This issues describes current state-of-the-art understanding regarding the dual diagnosis of TBI and pain.

The first article, by Drs Nicholson and Martelli, describes the "problem of pain" and gives a conceptual overview and framework for pain associated with cranial and cerebral trauma. This article reviews fundamental issues related to understanding the nature of pain, in particular chronic pain, and important distinctions between acute and chronic pain, neurobiological subtrates, the problem of mind-body dualism, and role of psychosocial factors and response bias.

Dr Walker reviews the pathophysiologic mechanisms involved in post-TBI pain disorders, examining both neurogenic and nonneurogenic contributors. Dr Ivanhoe and Hartman provide readers with an analysis of medical assessment and treatment of clinical pain disorders associated with TBI. They explore the broad spectrum of issues that may contribute to pain complaints in this patient population, as well as associated treatment methodologies. Drs Martelli, Zasler, Bender, and Nicholson then take on the task of reviewing and providing clinical caveats on the psychological, neuropsychological, and medical issues germane to the assessment and management of pain in persons with TBI. They present a biopsychosocial model for guiding assessment and treatment for the potentially widespread functional disability that accompanies chronic pain.

Wrapping up the clinical review articles for this special issue is an article addressing the integration of psychological and neuropsychological services in a multidisciplinary pain management treatment model for persons with TBI. This article is authored by Drs Branca and Lake and reviews the challenges that healthcare practitioners face in treating this set of dual diagnoses and the importance of utilizing a multidisciplinary team approach as well as adequate coordination of care across various healthcare disciplines. Lastly, a clinical research article and topic review article is presented by Dr Hecht that addresses occipital nerve blocks in post concussive headache.

It is our hope that this special issue of *JHTR* provides readers with a broad overview on the issues of pain assessment and management in persons with TBI. This foundation of knowledge will hopefully serve as impetus for readers in pursuing a broader clinical knowledge base and repertoire for optimizing the treatment of pain and related issues across the spectrum of TBI care.

—Nathan D. Zasler, MD, FAADEP, FAAPM&R, DAAPM, CIME
—Michael F. Martelli, PhD, DAAPM
Issue Editors

The Problem of Pain

Keith Nicholson, PhD; Michael F. Martelli, PhD

Pain problems, especially posttraumatic headache, are very common following head trauma. Pain may be the most significant problem, more disabling than any brain or other injuries, and interfering with aspects of cognition or other function. However, posttraumatic headache and most other chronic posttraumatic pain problems remain poorly understood. This article reviews fundamental issues that should be considered in understanding the nature of chronic pain including the distinction between acute and chronic pain; neurobiological distinctions between the lateral and medial pain system; nociceptive versus neuropathic or other central pain; sensitization effects; the widely accepted view of chronic pain as a multidimensional subjective experience involving sensory, motivational-affective and cognitive-behavioral components; the problem of mind-body dualism; the role of psychosocial factors in the onset, maintenance, exacerbation or severity of pain; plus issues of response bias and malingering.

Psychological, Neuropsychological, and Medical Considerations in Assessment and Management of Pain

Michael F. Martelli, PhD; Nathan D. Zasler, MD; Mark C. Bender, PhD; Keith Nicholson, PhD

Pain is a common yet challenging problem, particularly following traumatic injuries to the head or neck. It is a complex, multidimensional subjective experience with no clear or objective measures; yet it can have a significantly disabling effect across a wide range of functions. Persisting misconceptions owing to mind-body dualism have hampered advances in its understanding and treatment. In this article, a conceptualization of pain informed by recent research and derived from a more useful biopsychosocial model guides discussion of relevant medical, psychological, and neuropsychological considerations. This pain process model explains chronicity in terms of hyperresponsiveness and dysregulation of inhibitory or excitatory pain modulation mechanisms.

Related neurocognitive effects of chronic pain are examined and recommendations for minimizing its confounding effects in neuropsyhological evaluations are offered. A biopsychosocial assessment model is presented to guide understanding of the myriad of factors that contribute to chronicity. A brief survey of general classes and samples of the more useful pain assessment instruments is included. Finally, this model offers a rational means of organizing and planning individually tailored pain interventions, and some of the most useful pharmacologic, physical, and behavioral strategies are reviewed.

Clinical Caveats on Medical Assessment and Treatment of Pain After TBI

Cindy B. Ivanhoe, MD; Eric T. Hartman, MD

The diagnosis and management of pain in the patient with traumatic brain injury (TBI) can be difficult in light of the limitations imposed by the cognitive, language, and behavioral deficits. With patients in the acute rehabilitation setting, one must be vigilant for the often subtle signs and symptoms of pain. Causes more commonly seen in the population with TBI as a *consequence* of the injury itself include dysautonomia, neuropathic pain, spasticity, and heterotopic ossification. Headaches may be a consequence of TBI or associated with it for other reasons. Sources of pain *associated* with TBI include deep venous thrombosis and others. The reader is reminded that patients with TBI are subject to all the causes of pain that affect the general population.

Psychological and Neuropsychological Integration in Multidisciplinary Pain Management After TBI

Barbaranne Branca, PhD, ABPN, DABFE;
Alvin E. Lake III, PhD

The intersection of traumatic brain injury and posttraumatic chronic pain poses a significant challenge for the health practitioner. Effective intervention requires psychological and neuropsychological evaluation, multidisciplinary teamwork, and an understanding of a wide range of pain disorders and their relationship to traumatic brain injury. Assessment must include documentation of both current functioning and premorbid history. Pain interacts with cognitive impairment, mood and anxiety disorders, dysinhibition syndromes, and personality disorders, posing significant diagnostic dilemmas and treatment challenges. Coordinated care requires multiple, ongoing interventions from a variety of specialists. Patient involvement, focusing on internal locus of control, mediates successful treatment.

Occipital Nerve Blocks in Postconcussive Headaches

A Retrospective Review and Report

of Ten Patients

Jeffrey S. Hecht, MD

Headaches are common following traumatic brain injuries of all severities. Pain generators may be in the head itself or the neck. Headache assessment is discussed. Diagnosis and treatment of cervical headaches syndromes and, in particular, occipital neuralgia are reviewed. Finally, a retrospective study of 10 postconcussive patients with headaches who were treated with greater occipital nerve blocks is presented. Following the injection(s), 80% had a "good" response and 20% had a "partial" response. Occipital nerve block is a useful diagnostic and treatment modality in the setting of postconcussive headaches.

Pain Pathoetiology After TBI

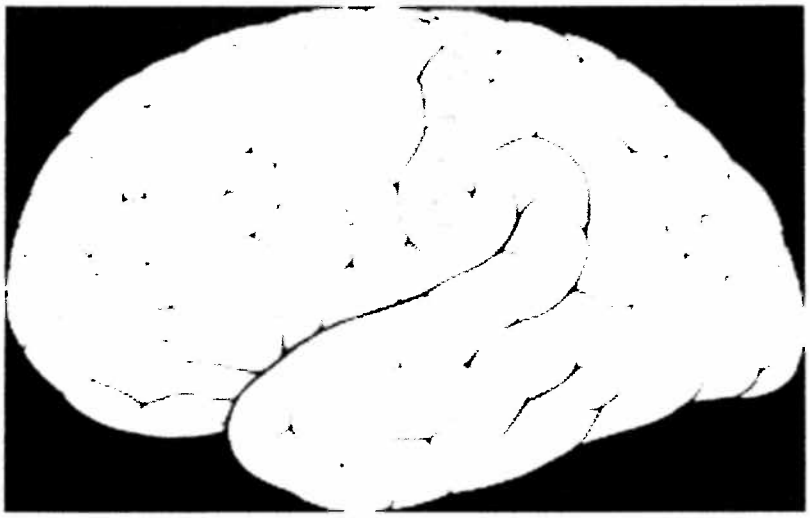
Neural and Nonneural Mechanisms

William C. Walker, MD

Individuals recovering from traumatic brain injury (TBI) frequently experience acute and chronic pain. Their pain experience is the net effect of many interacting and very complex physiologic, biochemical, and psychological mechanisms involving both the peripheral and central nervous system. This article reviews the basics of neural mechanisms and pathways of pain after TBI, and discusses clinical implications. Numerous intracranial and extracranial tissues must be considered in the evaluation of pain after TBI, with the specific mechanism of trauma influencing the anatomic distribution of injuries. The differential diagnosis usually falls into one of the following pathoetiologic classifications: primary or secondary musculoskeletal, vascular, visceral, and neural pain syndromes.

TEASER...

"Finally, the standards for specialty knowledge and training for treating pain parallel those for treating brain injury.[91] Similar expectancies apply to both, and neither should be compromised. At the core of all bioethical principles is the avoidance of harm. Virtually every ethics code issued by every healthcare profession and specialty enjoins its members to avoid doing harm by not practicing outside the limitations of their competence. Consistent with recent revisions and current ethical principles in medicine and psychology,[92–94] available options for brain injury specialists without specialized training and experience in pain management include referral to a professional with specialty competence; consultation with such specialists when referrals cannot be made; and acquisition of knowledge, supervision and training as indicated. When professions provide pain management services in the absence of specialty competence and without availing themselves of these options, they have ethical obligations to represent these limitations through tentativeness in opinions and conclusions and with complete transparency about the potential effects of these limitations."



White matter is one of the three main solid components of the central nervous system designated by color. The other two are gray matter and substantia nigra.

Structure

White matter is composed of myelinated nerve cell processes, or axons, which connect various gray matter areas (the locations of nerve cell bodies) of the brain to each other, and carry nerve impulses between neurons.

Cerebral- and spinal white matter do not contain dendrites, which can only be found in gray matter along with neural cell bodies, and shorter axons.[citation needed]

Function

The white matter is the tissue through which messages pass between different areas of gray matter within the nervous system. Using a computer network as an analogy, the gray matter can be thought of as the actual computers themselves, whereas the white matter represents the network cables connecting the computers together. The white matter is white because of the fatty substance (myelin) that surrounds the nerve fibers (axons). This myelin is found in almost all long nerve fibers, and acts as an electrical insulation. This is important because it allows the messages to pass quickly from place to place.

The brain in general (and especially a child's brain) can adapt to white-matter damage by finding alternative routes that bypass the damaged white-matter areas, and can therefore maintain good connections between the various areas of gray matter.

Unlike gray matter, which peaks in development in a person's twenties, the white matter continues to develop, and peaks in late middle age.[citation needed]

Location

White matter forms the bulk of the deep parts of the brain and the superficial parts of the spinal cord. Aggregates of grey matter such as the basal ganglia (caudate nucleus, putamen, globus pallidus, subthalamic nucleus, nucleus accumbens) and brain stem nuclei (red nucleus, substantia nigra, cranial nerve nuclei) are spread within the cerebral white matter.

The cerebellum is structured in a similar manner as the cerebrum, with a superficial mantle of cerebellar cortex, deep cerebellar white matter (called the "arbor vitae") and aggregates of grey matter surrounded by deep cerebellar white matter (dentate nucleus, globose nucleus, emboliform nucleus, and fastigial nucleus). The fluid-filled cerebral ventricles (lateral ventricles, third ventricle, cerebral aqueduct, fourth ventricle) are also located deep within the cerebral white matter.

Study Finds Loss of Small Nerve Fibers in Complex Regional Pain Syndrome (CRPS)
For release: Friday, May 19, 2006

A new study shows that a reduction in small-diameter nerve fibers is evident in the devastating chronic pain syndrome known as complex regional pain syndrome-type I (CRPS-I), which was formerly known as reflex sympathetic dystrophy. This finding of nerve damage could provide a biomarker, or aspecific physical trait, that clinicians could use in the future to help diagnose and measure the natural history ofCRPS.

The study of human skin biopsies provides some of the first evidence of a physical abnormality underlying CRPS-I. The results are published in the February issue of the journal Pain* and were funded in part by the National Institute of Neurological Disorders and Stroke (NINDS).

CRPS develops when a portion of the nervous system that normally conducts pain signals becomes electrically overactive. This phenomenon results in spontaneous pain. In the past, many doctors believed that this mysterious pain syndrome was a psychosomatic illness. CRPS type II, once known as causalgia, refers to pain after a nerve injury. CRPS-type I, formerly known as reflex sympathetic dystrophy (RSD), occurs in patients most often after trauma, but without clear nerve injury. Pain and other symptoms persist long after the initial injury has healed. The chronic pain of CRPS-I may emerge following a cut, burn, fracture, sprain, infection, surgery, or arthritis. Coronary artery disease and stroke may also be associated with onset of the disorder. Unlike conditions such as stroke, CRPS has no known risk factors and can affect anyone. The patient's arms or legs are usually involved, but CRPS may affect any part of the body. Patients also have other changes in the pain site such as swelling or changes in skin color

"CRPS is not really a disease process but a symptom complex. It has been difficult to accurately identify CRPS patients," says lead author Anne Louise Oaklander, MD, PhD, from Massachusetts General Hospital and Harvard Medical School in Boston. "However, absence of proof is not the proof of absence. With CRPS a great cross-section of people can be affected, since even drawing blood

can bring it on. The person can look absolutely fine on the outside but still can suffer from devastating pain," says Dr. Oaklander.

Currently, cases of CRPS are hard to prove or disprove. "It is difficult to see a patient who appears to be in distress, particularly if it's severe, and who's pleading with you to help, when you don't quite know what to do, ," Dr. Oaklander says. "Patients want an objective cause for their symptoms to be identified."

The typical neurology tests used to diagnose nerve injuries involve electromyography (EMG) and nerve conduction techniques that measure major motor or sensory nerve changes. Unfortunately the fiber nerve injuries in CRPS are not detected by this standard exam, which only measures large-fiber function. The difficulty in measuring small-fiber damage led Dr. Oaklander and her colleagues to propose skin biopsy for detection of CRPS. Skin biopsy, a procedure in which a sample of skin tissue is removed and examined, provides a very sensitive method for detecting small nerve fiber damage. "Skin biopsies are a window into the peripheral nervous system," says Dr. Oaklander.

In the study, small skin samples were taken from 18 adults with CRPS-I and seven people who had chronic pain from osteoarthritis, but not CRPS. Each subject identified the location of his or her maximum pain, a nearby symptom-free area on the same limb, and a pain free area on the opposite limb. Skin biopsies were taken from all three spots and the density of tiny projections extending from each nerve cell - small nerve fibers, or neurites - was measured. The results showed a decrease in the number of neurites in the CRPS-affected regions only. On average, about 30 percent of the neurites were missing in the affected limbs. The loss of neurites may cause the pain of CRPS by triggering a hyper-response on the part of the remaining neurons.

The next step for the researchers will be to create and test animal models that replicate the unique features of CRPS. This animal model may identify other biomarkers for the presence of the pain in CRPS patients and could improve medical care for patients with CRPS by providing the first and most important step of identification. In the meantime, the evidence now suggests that CRPS is a classifiable neurologic disorder. It also guides CRPS patients to seek treatments appropriate for nerve injury.

The NINDS is a component of the National Institutes of Health (NIH) in Bethesda, Maryland, and is the nation's primary supporter of biomedical research on the brain and nervous system. The NIH is comprised of 27 Institutes and Centers and is a component of the U. S. Department of Health and Human Services. It is the primary Federal agency for conducting and supporting basic, clinical, and translational medical research, and investigates the causes, treatments, and cures for both common and rare diseases. For more information about NIH and its programs, visit http://www.nih.gov.

*Oaklander AL, Rissmiller JG, Gelman LB, Zheng L, Chang Y, Gott R. "Evidence of focal small-fiber axonal degeneration in complex regional pain syndrome-I (reflex sympathetic dystrophy)." Pain, February 2006, Vol. 120, pp. 235-243.

-By Michelle D. Jones-London, Ph.D.

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BRAIN

White Matter Abnormalities in Mild Traumatic Brain Injury: A Diffusion Tensor Imaging Study

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BACKGROUND AND PURPOSE: Traumatic axonal injury is a primary brain abnormality in head trauma and is characterized by reduction of fractional anisotropy (FA) on diffusion tensor imaging (DTI). **Our hypothesis was that patients with mild traumatic brain injury (TBI) have widespread brain white matter regions of reduced FA involving a variety of fiber bundles and show fiber disruption on fiber tracking in a minority of these regions.**

MATERIALS AND METHODS: Ethics committee approval and informed consent were obtained. Twenty-one patients with mild TBI were investigated (men:women, 12:9; mean age ± SD, 32 ± 9 years). In a voxel-based comparison with 11 control subjects (men:women, 8:3; mean age, 37 ± 9 years) using z score analysis, patient regions with abnormally reduced FA were defined in brain white matter. MR imaging, DTI, and fiber tracking characteristics of these regions were described and analyzed using Pearson correlation, linear regression analysis, or the {chi}2 test when appropriate.

RESULTS: Patients had on average 9.1 regions with reduced FA, with a mean region volume of 525 mm3, predominantly found in cerebral lobar white matter, cingulum, and corpus callosum.

These regions mainly involved supratentorial projection fiber bundles, callosal fibers, and fronto-temporo-occipital association fiber bundles. Internal capsules and infratentorial white matter were relatively infrequently affected. Of all of the involved fiber bundles, 19.3% showed discontinuity on fiber tracking.

CONCLUSION: Patients with mild TBI have multiple regions with reduced FA in various white matter locations and involving various fiber bundles. A minority of these fiber bundles show discontinuity on fiber tracking.

Anatomy

The somatosensory system is spread through all major parts of a mammal's body (and other vertebrates). It consists both of sensory receptors and sensory (afferent) neurones in the periphery (skin, muscle and organs for example), to deeper neurones within the central nervous system.

General somatosensory pathway

A somatosensory pathway typically has three long neurons[1]: primary, secondary and tertiary (or first, second, and third).

* The first neuron always has its cell body in the dorsal root ganglion of the spinal nerve (if sensation is in head or neck, it will be the trigeminal nerve ganglia or the ganglia of other sensory cranial nerves).
* **The second neuron has its cell body either in the spinal cord or in the brainstem.** This neurones ascending axons will cross (decussate) to the opposite side either in the spinal cord or in the brainstem. **The axons of many of these neurones terminate in the thalamus (for example the ventral posterior nucleus, VPN), others terminate in the reticular system or the cerebellum.**
* In the case of touch and certain types of pain, **the third neuron has its cell body in the VPN of the thalamus and ends in the postcentral gyrus of the parietal lobe.**

Periphery

In the periphery, the somatosensory system detects various stimuli by sensory receptors, e.g. by mechanoreceptors. The sensory information (touch, pain, temperature etc.,) is then conveyed to the central nervous system by afferent neurones. There are a number of different types of afferent neurones which vary in their size, structure and properties. Generally there is a correlation between the type of sensory modality detected and the type of afferent neurone involved. So for example slow, thin unmyelinated neurones conduct casual touch whereas faster, thicker, myelinated neurones conduct pain.

Spinal cord

In the spinal cord, the somatosensory system [2] includes ascending pathways from the body to the brain. One major target within the brain is the postcentral gyrus in the cerebral cortex. This is the target for neurones of the Dorsal Column Medial Lemniscal pathway and the Ventral Spinothalamic pathway. **Note that many ascending somatosensory pathways include synapses in either the thalamus or the reticular formation before they reach the cortex. Other ascending pathways, particularly those involved with control of posture are projected to the cerebellum. These include the ventral and dorsal spinocerebellar tracts.** Another important target for afferent somatosensory neurones which enter the spinal cord are those neurones involved with local segmental reflexes.

Brain

The primary somatosensory area in the human cortex is located in the postcentral gyrus of the parietal lobe. The postcentral gyrus is the location of the primary somatosensory area, the main sensory receptive area for the sense of touch. Like other sensory areas, there is a map of sensory space called a homunculus at this location. For the primary somatosensory cortex, this is called the sensory homunculus. Areas of this part of the human brain map to certain areas of the body, dependent on the amount or importance of somatosensory input from that area. For example,

there is a large area of cortex devoted to sensation in the hands, while the back has a much smaller area. Interestingly, one study showed somatosensory cortex was found to be 21% thicker in 24 migraine sufferers, on average than in 12 controls[3], although we do not yet know what the significance of this is. Somatosensory information involved with proprioception and posture also targets an entirely different part of the brain, the cerebellum.

Physiology

Initiation of probably all "somatosensation" begins with activation of some sort of physical "receptor". These somatosensory receptors, tend to lie in skin, organs or muscle. The structure of these receptors is broadly similar in all cases, consisting of either a "free nerve ending" or a nerve ending embedded in a specialised capsule. They can be activated by movement (mechanoreceptor), pressure (mechanoreceptor), chemical (chemoreceptor) and/or temperature. In each case, the general principle of activation is similar; the stimulus causes depolarisation of the nerve ending and then an action potential is initiated. This action potential then (usually) travels inward towards the spinal cord.

Technology

The new research area of haptic technology can provide touch sensation in virtual and real environments. This new discipline has started to provide critical insights into touch capabilities.

[edit] See also

* Cell signalling
* Special senses
* Molecular Cellular Cognition
* Muscle spindle

Notes

1. ^ Saladin KS. Anatomy and Physiology 3rd ed. 2004. McGraw-Hill, New York.
2. ^ Nolte J.The Human Brain 5th ed. 2002. Mosby Inc, Missouri.

3. ^ "Thickening in the somatosensory cortex of patients with migraine." Alexandre F.M. DaSilva, Cristina Granziera, Josh Snyder, and Nouchine Hadjikhani. Neurology, Nov 2007; 69: 1990 - 1995.

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The lateral postcentral gyrus is a prominent structure in the parietal lobe of the human brain and an important landmark. It was initially defined from surface stimulation studies of Penfield, and parallel surface potential studies of Bard, Woolsey, and Marshall. Although initially defined to be roughly the same as Brodmann areas 3, 1 and 2, more recent work by Kaas has suggested that for homogeny with other sensory fields only area 3 should be referred to as "primary somatosensory cortex", as it received the bulk of the thalamocortical projection from the sensory input fields.